U.S. Department of Labor

Office of Administrative Law Judges
Seven Parkway Center - Room 290
Pittsburgh, PA 15220

(412) 644-5754
(412) 644-5005 (FAX)

Issue date: 28Sep2001

CASE NO.: 2001-BLA-116

In the Matter of:

SUSAN J. BRADLEY, surviving spouse of
RICHARD P. BRADLEY
Claimant

v.

GREENWICH COLLIERIES, INC.
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party in Interest

Appearances:

Robert J. Bilonick, Esq.
For the Claimant

John J. Bagnato, Esq.
For the Employer

Before: DANIEL L. LELAND
Administrative Law Judge

DECISION AND ORDER - DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq*. In accordance with the Act and the pertinent regulations, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as black lung.

A formal hearing was held in Ebensburg, Pennsylvania on June 13, 2001, at which all parties were afforded full opportunity to present evidence and argument, as provided by the Act and the regulations found in Title 20 Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title. At the hearing, Director's exhibits (DX) 1 - 37, Claimant's exhibits (CX) 1 - 4, and Employer's exhibits (EX) 1 - 8 were admitted into evidence. The record was held open for thirty days post hearing for Dr. Joshua A. Perper's deposition, which was marked as CX 5.

ISSUE

The only issue is whether the miner's death was due to pneumoconiosis. The employer has stipulated to thirty-seven years of coal mine employment.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History and Factual Background

Claimant is the widow of Richard P. Bradley (The miner or Decedent), who was born on March 6, 1926 and died on May 16, 1999. (DX 1) The miner initially filed a claim on July 19, 1989 and was awarded benefits by the district director on October 26, 1989. (DX 35-1, DX 35-27) Employer appealed and the undersigned denied benefits on January 29, 1991. (DX 35-46) Claimant filed the instant claim for benefits on August 27, 1999. (DX 1) The district director denied benefits by Proposed Decision and Order Memorandum of Conference on August 22, 2000. (DX 32) The instant case was referred to the Office of Administrative Law Judges for a hearing on September 29, 2000. (DX 37)

Claimant testified that the miner retired from the mines in 1988 due to breathing problems. (TR 10) The miner's course of treatment for his breathing problem consisted of inhalers, home oxygen, and the medications of Theo-Dur, Proventil, and Atrovent. (TR 10-11) Claimant also testified that when the miner would exert himself he would get out of breath and have to sit down. (TR 12) The miner was admitted at Miner's Hospital for his breathing problem and then transferred to Haida Manor, where he subsequently died. Id. The miner smoked cigarettes until 1988 or 1989. (TR 13) He would smoke approximately one-half pack to one pack per day, however, he cut back to two cigarettes per day in 1988. Id.

Medical Evidence

The summary of x-ray evidence in the undersigned's Decision and Order - Denying Benefits of January 29, 1991 is incorporated by reference.

The miner's death certificate, dated May 16, 1999, listed the causes of death as the following: severe chronic obstructive lung disease; chronic hypoxia; chronic renal failure; and coronary artery disease. (DX 11) The death certificate was signed by Dr. Matthew J. Dvorchak.

The autopsy report was prepared by Dr. Curtis S. Goldblatt, who is board certified in anatomic and clinical pathology. (DX 12) Gross examination of the lungs revealed anthracotic pigment staining over eighty percent of the surface, focally fibrotic lungs on palpation, and anthrocotic pigmentation involving fifty percent of the parenchyma. His microscopic examination revealed "severe architectural distortion of the right upper lobe with dense parenchymal fibrous scar formation more than two centimeters in greatest dimension, extending to the pleural surface." These fibrous scars contained anthrasilicotic pigment laden macrophages. Sections from other areas of the lung showed anthrasilicotic macule formation up to four millimeters occupying fifty percent of the parenchyma. Dr. Goldblatt concluded the miner's cause of death was acute bronchopneumonia with significant contributing causes of death of complicated coal workers' pneumoconiosis and severe pulmonary emphysema.

Dr. Joshua Perper, a board certified anatomic, clinical, and forensic pathologist, reviewed the miner's medical records and examined the autopsy slides on December 14, 1999. (DX 13) He noted that the miner worked thirty-seven years as an underground coal the miner and was a heavy smoker until he quit ten years prior to his death. Dr. Perper determined that the lung slides showed complicated coal workers' pneumoconiosis with areas of fibro-anthracosis slightly exceeding two centimeters on the background of severe simple coal workers' pneumoconiosis. Dr. Perper also found hypoxia associated with pneumoconiosis and chronic emphysema. The etiology of emphysema was cigarette smoking and coal workers' pneumoconiosis. He explained that although the miner's previous smoking history played an important role in his development of emphysema, this does not preclude the important role of coal dust exposure and pneumoconiosis. Dr. Perper explained that although some x-rays did not reveal significant pneumoconiosis, it is possible that at the time of the reading the pneumoconiotic changes were not sufficiently developed to allow detection or the radiologists may have missed the diagnosis of pneumoconiosis. Dr. Perper stated that pneumoconiosis was a substantial contributory cause of his death through the presence of complicated pneumoconiosis with clinical symptomatology, objectively substantiated hypoxemia, obstructive pulmonary deficits, and chronic obstructive pulmonary disease (COPD).

Dr. Stephen T. Bush, who is board certified in anatomic and clinical pathology and medical microbiology, analyzed the miner's autopsy slides and report. In his February 3, 2000 report (DX 21), Dr. Bush concluded that the miner had a mild degree of pneumoconiosis and centrilobular emphysema. He stated that the macules and micro nodules of pneumoconiosis, measuring between 0.1 and 0.4 centimeters, are too small to meet the requirements of a diagnosis of complicated pneumoconiosis. He

disagreed with Dr. Goldblatt's diagnosis of progressive massive fibrosis, stating that Dr. Goldblatt's diagnosis is a scar of the pleura and underlying lung from a past inflammatory process. Dr. Bush stated that the miner's cause of death was from bronchopneumonia associated with centrilobular emphysema. He determined that pneumoconiosis was too limited in degree and extent to have caused or contributed to the bronchopneumonia in any significant degree.

Dr. Richard L. Naeye, who is board certified in anatomic and clinical pathology, reviewed the medical records and autopsy slides and submitted a report dated March 4, 2000. (DX 22) He found mild to moderately severe coal workers' pneumoconiosis. The physician stated that the anthracotic macules and micronodules are too small to meet minimum requirements of diagnosis of complicated pneumoconiosis and he disagreed with Dr. Perper's findings that areas of fibro-anthracosis slightly exceed two centimeters. Dr. Naeye explained that the miner's x-ray readings of 0/0 and 0/1 after retiring from the coal mines in 1987 indicates that his pneumoconiosis must have been mild because when pneumoconiosis is moderate to severe the lesions are visible on x-ray. He explained that the dichotomy between the negative x-ray readings and what was found in the autopsy tissues suggests that the autopsy tissues removed for microscopic examination are not representative of the lungs as a whole. Dr. Naeye attributed the miner's small airways obstruction to cigarette smoking and determined that death was due to severe, acute lobular pneumonia and renal failure. He opined that the miner's simple coal workers' pneumoconiosis was too mild to have made a measurable contribution to his respiratory disability or to have hastened his death.

Dr. Bush reviewed additional slides and medical documents on March 21, 2000. (DX 25) Although the additional autopsy slides revealed a greater severity of pneumoconiosis than Dr. Bush originally diagnosed in his February 3, 2000 report, the presence of pneumoconiosis destroyed no more than five percent of lung tissue and therefore the disease is mild rather than severe. He determined that the miner's death resulted from acute bronchopneumonia associated with moderately severe centrilobular emphysema, which is typical of the emphysema noted in heavy cigarette smokers. Dr. Bush found no causal relationship between the simple coal workers' pneumoconiosis or coal mine dust exposure and death from pneumonia and chronic obstructive pulmonary disease. The physician determined that the miner would have died at the same time and in the same manner had he had never been a coal the miner. He read Dr. Perper's December 15, 1999 report and disagreed with the physician's diagnosis of complicated pneumoconiosis. Dr. Bush explained that the lesion exceeding two centimeters is a subpleural post inflammatory scar which is trapped in a small amount of dust pigment, and not a coal worker macronodule.

Dr. Harvey Mendelow, who is board certified in anatomic and clinical pathology, reviewed the miner's medical records, diagnostic tests, and autopsy slides on June 20, 2000. (DX 29) He determined that the miner died as result of bilateral acute pneumonia and underlying severe COPD due to advanced emphysema and chronic bronchitis. Dr. Mendelow diagnosed mild to moderate pneumoconiosis, but was in disagreement with Drs. Goldblatt and Perper's diagnosis of complicated coal workers' pneumoconiosis and progressive massive fibrosis. The physician stated these areas represent previous acute and chronic lung inflammation probably resulting from past episodes of

pneumonia. He explained that none of the miner's chest x-rays taken from his retirement until his death revealed complicated pneumoconiosis and that the pathologic findings at autopsy do not meet the accepted standards established for this diagnosis. Dr. Mendelow concluded that The miner would have died at the same time and in the same manner regardless if he had been a the miner.

Dr. Gregory Fino, who is board certified in internal medicine and pulmonary diseases, reviewed the miner's medical records and submitted a report dated July 6, 2000. (DX 30) Dr. Fino found pathological evidence of simple pneumoconiosis. He concluded that the miner died as a result of chronic respiratory disease due to smoking and that coal mine dust did not contribute to his death. Dr. Fino determined that the miner would have died when he did had he never been a coal the miner.

Dr. Naeye reviewed three autopsy slides and submitted a report dated July 10, 2000. (DX 31) He explained that if these three slides were representative of the lungs as a whole the appropriate diagnosis would be severe, simple coal workers' pneumoconiosis. Dr. Naeye further explained that the other twelve slides had macules and micro nodules that were smaller and fewer in number than these three slides. Furthermore, if the three slides were representative of the lungs as a whole the x-rays taken of the miner's lungs would have revealed moderately severe or severe coal workers' pneumoconiosis; however, all of the miner's x-rays have been interpreted as 0/0 or 0/1. Additionally, Dr. Naeye stated "{a}s far as CWP is concerned these older x-ray findings were valid on the day this man died because simple CWP does not progress after a worker quits mining coal."

Dr. Waheeb Rizkalla, who is board certified in anatomic and clinical pathology, analyzed the miner's medical records and autopsy slides on December 13, 2000. (CX 1) He stated that the miner's microscopic slides revealed anthrasilicotic deposits in the lung tissue with silica crystals in the form of macules and macronodules and scar emphysema. The immediate cause of death was severe acute bronchopneumonia which was affected by the coal dust and coal workers' pneumoconiosis in the form of macules, macronodules, and focal dust emphysema. The physician opined that pneumoconiosis accelerated the miner's death. Dr. Rizkalla disagreed with the physicians who attributed the miner's pulmonary impairment solely to cigarette smoking. He explained that smoking does not induce focal emphysema; however, centrilobular emphysema is induced by both smoking and coal dust exposure.

Dr. Philip T. Cagle, a board certified anatomic and clinical pathologist, reviewed the miner's autopsy slides on January 2, 2001. (EX 1) He determined that the lung slides showed scattered macules and micronodules between 0.2 and 0.5 centimeters in diameter with focal emphysema totaling five percent of the lung surface area. The physician opined that the miner's cause of death was acute pneumonia. Dr. Cagle stated that the miner's bullous emphysema was caused by tobacco smoking and his focal emphysema surrounding the micronodules was not significant in terms of the amount of lung tissue involved. He determined that there was no causal relationship between acute pneumonia and the tobacco-caused bullous emphysema, which were the causes of the miner's death, and occupational pulmonary disease or dust exposure. Therefore, Dr. Cagle opined that the miner would have died at the same time and in the same manner whether or not he had been exposed to coal dust.

Dr. Rizkalla drafted a supplement letter dated January 12, 2001. (CX 3) He explained that he measured nodules seen to be on the average of seven millimeters, while other nodules are more than one centimeter in greatest dimension with areas of fibrosis measuring one centimeter to one and one-half centimeter with anthracoid pigmentation. Dr. Rizkalla explained that medical literature defines complicated pneumoconiosis any where from one to three centimeters in dimension. The nodules found in the miner's lungs measuring more than one centimeter in greatest dimension and areas of anthrasilicotic pigmentation with fibrosis up to one and one-half centimeters in dimension qualifies under medical literature as complicated pneumoconiosis.

The autopsy slides and medical records were reviewed by Dr. Joseph Tomashefski, a board certified anatomic and clinical pathologist. In a February 9, 2001 report (EX 2), Dr. Tomashefski determined that the lung slides showed macules and micronodules ranging from one millimeter to seven millimeters in dimension occupying about five to fifteen percent of the lung tissue represented on the slides. He opined that the miner suffered from moderately severe simple coal workers' pneumoconiosis, along with severe end-stage mixed panacinar and centriacinar emphysema, chronic bronchitis, and severe acute diffuse pneumonia. Because the largest nodular lesion measured only seven and two tenths millimeters, Dr. Tomashefski determined that the miner did not have complicated pneumoconiosis because that diagnosis requires nodules of at least two centimeters in size. The physician further stated that the miner's pneumoconiosis was a very minor component of his overall lung remodeling, thus neither causing nor contributing to his death. Dr. Tomashefski based this opinion on the x-ray evidence, which indicated that the miner's simple coal workers' pneumoconiosis was not severe enough to have caused him any significant respiratory impairment when he was alive. The physician also stated that the miner's advanced emphysema was entirely due to cigarette smoke exposure. He further opined that the miner's chronic bronchitis was not due to coal dust exposure or coal mine employment because his chronic bronchitis had persisted for years after his coal dust exposure had ceased.

Dr. Rizkalla was deposed on March 13, 2001. (CX 4) He stated that the microscopic slides of the lung tissue were technically acceptable in quality and quantity and the physician found these slides to be representative of the disease process in the lungs. (CX 4, pp. 8-9) He explained that a random selection of the lungs is based on taking different sections of the lungs without stressing upon a specific area. (CX 4, p. 15) Although this process may result in the slides varying in the amount of the disease process, this does not imply that the slides are not representative of the disease process. (CX 4, p. 16) Dr. Rizkalla explained that the slides revealed two disease processes, coal workers' pneumoconiosis and bronchopneumonia. (CX 4, p. 9) He diagnosed severe coal workers' pneumoconiosis based upon the number of macronodules contained in the slides, the distribution of the nodules in the lungs, and reading the original autopsy report. (CX 4, p. 14) Dr. Rizkalla reiterated his previous diagnosis of emphysema and explained that focal emphysema is not caused by smoking while centrilobular emphysema is caused by both smoking and pneumoconiosis. (CX 4, pp. 18-19) The physician stated that he cannot exclude cigarette smoking from the etiology of the miner's centrilobular emphysema because the miner had a significant smoking history and it is medically accepted that smoking causes this form of emphysema. (CX 4, p. 24-25) Nor could the physician exclude the miner's lengthy coal

mine employment as the cause of his emphysema. (CX 4, p. 25) He reiterated his previous conclusion that smoking and coal workers' pneumoconiosis led to the miner's pulmonary impairment, which contributed to his death. (CX 4, p. 27, 35)

Dr. Steven P. Griffin, who is board certified in anatomic and clinical pathology, examined the miner's medical records and autopsy slides. (EX 3) The slides revealed a mild extent of simple coal workers' pneumoconiosis, which the physician attributed to the miner's coal dust exposure. Dr. Griffin disagreed with the diagnosis of complicated pneumoconiosis for the following reasons: the fibrous areas measuring up to two centimeters referred in the autopsy is subpleural scarring and not lesions of pneumoconiosis; and the x-ray interpretations do not support a diagnosis of complicated pneumoconiosis. He concluded the miner's cause of death as extensive acute pneumonia in addition to bullous and centrilobular emphysema, indicating a significant element of COPD. Dr. Griffin opined that the miner's simple pneumoconiosis did not contribute to his death because simple pneumoconiosis does not cause pneumonia and did not have a measurable effect on his breathing ability. He stated that cigarette smoking is the etiology of the miner's emphysema and subsequent breathing distress. Dr. Griffin further stated that the miner would have died at the same date and time and in the same manner whether he had ever set foot in a coal mine.

In his addendum letter dated May 9, 2001, Dr. Fino commented on the article Airways obstruction, coal mining, and disability. (EX 4) He stated that the methodology of the study indicates that a majority of the coal the miners studied were referred by coal companies because when a the miner files a claim for black lung benefits the referral is part of the evaluation of the claim. Dr. Fino explained that this methodology did not flaw the results of the study.

Dr. Bush was deposed on January 10, 2001. (EX 5) He reiterated his previous opinion that The miner had a mild degree of simple coal workers' pneumoconiosis and severe centrilobular emphysema. (EX 5, p. 9) The physician further reiterated that the miner's centrilobular emphysema resulted from his heavy smoking history. Id. Dr. Bush attributed the miner's emphysema to smoking cigarettes rather than coal dust exposure because less than five percent of his lung tissue was damaged by pneumoconiosis while he had a severe degree of centrilobular emphysema. (EX 5, p. 10) He explained that the pneumoconiosis lesions were in some places incidentally randomly associated with centrilobular emphysema; however, in the majority of the lung areas was an independent process affecting the lung. Id. Dr. Bush further explained that the areas in the lung that show fibrotic change is the result of scarring from previous infections, and not severe pneumoconiosis as diagnosed by Drs. Goldblatt and Perper. (EX 5, pp.11-12) He concluded that the miner died from bronchopneumonia associated with centrilobular emphysema as a result of heavy cigarette smoking. (EX 5, p. 13) The physician reiterated his previous conclusion that there was no casual nexus between the miner's exposure to coal dust and his death. Id.

Dr. Fino was deposed on January 12, 2001. (EX 6) He determined that the miner died as a result of a chronic respiratory disease with superimposed pneumonia. (EX 6, p. 7) The physician explained that the degree of clinical pneumoconiosis in the miner's lungs as insufficient to cause any respiratory impairment. (EX 6, p. 10) It is Dr. Fino's opinion that the miner's disability and subsequent death was a complication of smoking and that coal mine dust played a negligible role in either his pulmonary disability or death. (EX 6, p. 14)

Dr. Naeye was deposed on February 23, 2001. (EX 7) He averred that pneumoconiosis only affected two or three percent of the miner's total lung parenchyma and the largest legion caused by coal dust exposure measured four millimeters in diameter. (EX 7, pp. 17-18) Dr. Naeye repeated his earlier statement that the miner also suffered from both focal and centrilobular emphysema, with the focal emphysema only constituting a few percent of the miner's total emphysema, chronic bronchitis, and bronchiolitis. (EX 7, pp. 18-19) He opined that the miner's coal dust exposure was slightly related to his death insofar that coal dust exposure originally contributed to his chronic bronchitis and bronchiolitis; however, these disorders persisted after the miner left the coal mines because of his heavy smoking history. (EX 7, pp. 22-24) Dr. Naeye repeated his earlier statement that the slides were not representative of the miner's lungs as a whole because if he had suffered from moderately severe pneumoconiosis the macules and micronodules would be of sufficient size and number to produce lesions that are easily visible on an x-ray. (EX 7, pp. 46-47) The physician opined that the large zones of fibrosis in the miner's lung were old areas of pneumonia and not related to coal workers' pneumoconiosis. (EX 7, pp. 47-48)

In his deposition of June 5, 2001, Dr. Cagle repeated his earlier statement that cause of death was acute pneumonia with severe bullous emphysema due to cigarette smoking. (EX 8, p. 31) He explained that the miner's bullous emphysema made him susceptible to pneumonia. (EX 8, p. 32) The miner's coal workers' pneumoconiosis was not a contributing factor in his development of pneumonia. (EX 8, pp. 32-33) Dr. Cagle did not find pathological evidence of complicated pneumoconiosis or progressive massive fibrosis. (EX 8, p. 33)

Dr. Perper was deposed on July 2, 2001. (CX 5) He stated that the miner had diagnostic evidence of lung disease since 1987 and it progressed over the years. (CX 5, pp. 7, 8, 11) Dr. Perper found that the microscopic slides were of good quality and representative of the lungs as a whole. (CX 5, p. 12) He discounted Dr. Naeye's opinion that because the x-ray evidence did not correlate with the tissue slides, the slides were not representative of the lungs as a whole. (CX 5, p. 13) Dr. Perper explained that the only indicator of a representative sampling of tissue is whether the sampling of tissue was taken from those areas of the lung which shows various areas of injury and normal tissue; the representativeness of the area is not a function of the radiological findings. Id. While Dr. Perper averred that there were areas of acute bronchopneumonia found in the miner's lungs, he did not believe that there were areas of organized pneumonia. (CX 5, pp. 21-22) Dr. Perper opined that the miner's cause of death was pulmonary impairment with coal workers' pneumoconiosis, with associated

centrilobular emphysema contributed by smoking. (CX 5, p. 23) The physician explained that the miner's significant smoking history and pneumoconiosis both contributed to his lung disease, and therefore a person cannot exclude one while favoring the other. (CX 5, p. 24)

Conclusions of Law

Claimant has the burden of proving by a preponderance of the evidence that the miner's death was due to pneumoconiosis which arose out of coal mine employment. § 718.205(a). Death will be considered due to pneumoconiosis where the medical evidence establishes that the miner's death was due to pneumoconiosis, or where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or where the presumption in § 718.304 is applicable. § 718.205(c). Under amended § 718.205(c), pneumoconiosis is a "substantially contributing cause" of a the miner's death if it hastens the miner's death. § 718.205(c)(5).

Section 718.304 of the Act creates an irrebuttable presumption of total disability due to pneumoconiosis and death due to pneumoconiosis if the evidence establishes that a miner is suffering from a chronic dust disease of the lung which is manifested by (a) x-ray opacities greater than one centimeter in diameter and classified as Category A, B, C; or (b) an autopsy or biopsy which yields massive lesions in the lung; or (c) when diagnosed by means other than (a) or (b), would be a condition which could reasonably be expected to yield the results described in (a) or (b). 20 C.F.R. § 718.304. The administrative law judge must consider and weigh all relevant evidence in determining whether the evidence is sufficient to establish the presence of complicated pneumoconiosis. *Melnick v. Consolidation Coal Co.*, 16 BLR 1-31 (1991) (ALJ must first evaluate the evidence in each category and then weigh the evidence together prior to invocation); *Maypray v. Island Creek Coal Co.*, 7 BLR 1-683 (1985).

There are twenty-two interpretations of four different x-ray films contained in the record. Although seven were read positive for pneumoconiosis, none of these seven readings were interpreted as showing large opacities. I note that the most recent x-ray interpretation of record is dated June 25, 1990, approximately nine years prior to the miner's death. Since pneumoconiosis is a latent and progressive disease, the x-ray interpretations of record are not very probative as to the miner's degree of pneumoconiosis on the date of his death. The preponderance of the x-ray evidence is negative for complicated pneumoconiosis and does not invoke the irrebuttable presumption at § 718.304(a).

Dr. Fino is the only physician of record who is not a board certified pathologist. I find that he is not as qualified as the board certified pathologists in diagnosing complicated pneumoconiosis pathologically and thus accord his opinion little weight. Dr. Naeye believes that simple coal workers' pneumoconiosis does not progress after cessation of mining. (DX 31) This statement repudiates the progressive nature of pneumoconiosis as defined by statute and case law. *See 20 C.F.R. § 718.201(c)* (pneumoconiosis is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.); *Labelle Processing Co. v. Swarrow*, 72 F.3d

308, 314(3rd Cir. 1995); *Mullins Coal Co., Inc. v. Director, OWCP*, 484 U.S. 135, 151 (1987). Furthermore, Dr. Naeye stated that the dichotomy between the negative x-ray readings and the findings at autopsy suggests that the slides were not representative of the lungs as a whole. Dr. Naeye is the only pathologist of record to call into question the validity of the slides; both Drs. Rizkalla and Perper refuted Dr. Naeye's opinion and gave a detailed explanation of how these slides were technically acceptable and representative of the lungs as a whole. For these reasons I find Dr. Naeye's opinion unreliable and give his opinion little weight.

Drs. Bush, Mendelow, and Tomashefski all agreed that the macules and micronodules of pneumoconiosis measuring between one and seven millimeters are too small to meet the requirements of complicated pneumoconiosis. Drs. Bush, Mendelow, and Griffin believed that the lesion measuring one and one-half centimeters is a subpleural scar and not a lesion of progressive massive fibrosis. Dr. Bush explained that the lesion is a post-inflammatory scar which is trapped in a small amount of dust pigment. Furthermore, Drs. Mendelow, Tomashefski, and Griffin discounted complicated pneumoconiosis on the basis that the miner's chest x-rays did not reveal complicated pneumoconiosis. Dr. Cagle found no pathological evidence of complicated pneumoconiosis or progressive massive fibrosis.

On microscopic examination, Dr. Goldblatt, the autopsy prosector, found "dense parenchymal fibrous scar formation more than two centimeters in greatest dimension, extending to the pleural surface" containing anthrasilicotic pigment laden macrophages. He also listed complicated coal workers' pneumoconiosis as a significant contributing cause of death. Dr. Perper reviewed the slides and found complicated coal workers' pneumoconiosis with areas of fibro-anthracosis exceeding two centimeters in diameter. While Dr. Rizkalla did not specifically diagnose complicated pneumoconiosis, he explained that the lesions measuring over one centimeter in dimension qualify as complicated pneumoconiosis.

The disagreement over whether the miner had complicated pneumoconiosis centers on two issues. First, whether the macules and micronodules of pneumoconiosis constitute complicated pneumoconiosis, taking into account the size of the lesions. Second, whether the area measuring up to two centimeters in diameter is progressive massive fibrosis or a subpleural scar from a previous inflammation.

The Board has held that an administrative law judge properly found the irrebuttable presumption pursuant to § 718.304(b) where the physician diagnosed complicated pneumoconiosis, describing the lesions as up to one centimeter in diameter. *Gruller v. Bethenergy Mines, Inc.*, 16 BLR 1-3 (1991). The Third Circuit, the circuit in which this case arises, has affirmed the finding of complicated pneumoconiosis where the physician described nodules measuring between one and one and one-half centimeters in diameter. *Clites v. J. & L. Steel Corp.*, 663 F.2d 14 (3rd Cir. 1981). Case law has made it clear that a diagnosis of complicated pneumoconiosis is proper where the nodules measure less than two centimeters in diameter. Although Drs. Perper and Rizkalla reported pneumoconiosis nodules measuring over one centimeter, the preponderance of the medical evidence reveals that the largest nodule found on the miner's lungs measured a little over seven millimeters in

diameter. I have found no case law that supports a finding of complicated pneumoconiosis where the lesions were less than one centimeter in diameter. Therefore I find that the pneumoconiosis lesions measuring up to seven millimeters in diameter do not constitute a finding of complicated pneumoconiosis under § 718. 304(b).

As for the diagnosis of the lesions located in the upper right lobe, each party presented a significant amount of medical reports and depositions in support of its position. In such cases, it is the task of an administrative law judge to accept one side's testimony over the testimony of the other. *Walker v. Universal Terminal & Stevedoring Corp.*, 645 F.2d 170 (3rd Cir. 1981). All of the pathologists are board certified in anatomic and clinical pathology and possess superior credentials; however, I find the opinion of Drs. Mendelow, Tomashefski, and Griffin more credible than the opinions of Drs. Goldblatt, Perper, and Rizkalla. Although Dr. Goldblatt, the autopsy prosector, noted a lesion consistent with progressive massive fibrosis and determined that the miner's death was significantly contributed to by complicated coal workers' pneumoconiosis, he failed to explain how complicated pneumoconiosis contributed to the miner's death. Dr. Goldblatt's diagnosis of progressive massive fibrosis was based upon the microscopic examination, and not the gross examination, of the lung tissue. Since Dr. Goldblatt did not rely on his gross examination of the body, his opinion is not entitled to deference because he is the autopsy prosector. *See Urgolites v. Beth Energy Mines*, 17 BLR 1-20, 1-23 (1992). Although Dr. Perper determined that the lesions were consistent with progressive massive fibrosis, he also found that the miner suffered from hypoxia. He is the only pathologist of record to make this diagnosis and the record is void of any evidence that the miner suffered from hypoxia. Dr. Perper's diagnosis of hypoxia detracts from the credibility of his opinion as a whole and I accord it little weight. Drs. Mendelow, Tomashefski, Griffin, and Bush all determined that the lesions were a result of a previous infection such as pneumonia rather than pneumoconiosis. Each gave a well reasoned opinion and each opinion is supported by the objective evidence of record. Weighing all of the evidence under § 718.304(b), I find that the preponderance of the evidence is not sufficient to invoke the irrebuttable presumption.

Section 718.304(c) provides that the irrebuttable presumption is established by other means than those specified in paragraphs (a) or (b). There is no CT evidence and all of the medical reports have been summarized above. There is no evidence of record which falls under § 718.304(c). [1] Hence Claimant is unable to invoke the irrebuttable presumption of § 718.304 through evidence under § 718.304(c).

After applying the standard in each prong, an administrative law judge must perform an equivalency finding between the lesions and x-ray interpretations. *Clites v. Jones & Laughlin Steel Corp.*, 663 F.2d 14 (3rd Cir. 1981). Both the x-ray evidence and autopsy evidence are insufficient to invoke the presumption. Although I found that the x-ray interpretations of record were not probative of

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[1] If I were to include the physician opinions as evidence under paragraph (c), I would weigh them in the same manner.

the miner's condition at his death, the autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP*, 7 BLR 1-688 (1985). Therefore, weighing all of the evidence together I find that the preponderance of the evidence is insufficient to invoke the irrebuttable presumption that the miner's death was due to pneumoconiosis. I must now determine whether pneumoconiosis was a substantially contributing cause of the miner's death.

Dr. Goldblatt, the autopsy prosector, found that the miner's pneumoconiosis contributed to his death. Drs. Perper and Rizkalla both agreed that the etiologies of the miner's emphysema were cigarette smoking and coal dust exposure. They both explained that the miner's significant smoking history, along with extensive underground coal mine history, contributed to his lung disease and one cannot be excluded while favoring the other. Drs. Bush, Mendelow, Cagle, Tomashefski, and Griffin concluded that the miner's smoking history alone led to his pulmonary impairment, which contributed to his death. Drs. Bush, Tomashefski, and Griffin stated that the miner's pneumoconiosis, which destroyed approximately five percent of his lung tissue, was a minor component in his overall lung remodeling. Furthermore, Dr. Cagle opined that miner's focal emphysema was not significant in terms of the amount of lung tissue involved.

Dr Goldblatt did not explain how pneumoconiosis contributed to the miner's death and his opinion is therefore not well reasoned. While it is undisputed that the miner had an extensive underground coal mine history, the coal mine history alone is not sufficient for a finding that pneumoconiosis substantially contributed to the miner's death. Drs. Bush, Mendelow, Cagle, Tomashefski, and Griffin all considered the miner's extensive coal dust exposure, but concluded that the objective medical evidence indicates that the miner's pulmonary impairment was due to his cigarette smoking. I credit these thorough, well reasoned, opinions that the miner's pneumoconiosis did not contribute to his death in any manner. I conclude that pneumoconiosis did not cause, contribute, or hasten the miner's death.

As the evidence does not show that the miner's death was due to pneumoconiosis, the claim will be denied. In light of this denial, Claimant's counsel is precluded from charging a fee for his professional services.

ORDER

IT IS ORDERED that the claim of Susan J. Bradley, widow of Richard P. Bradley, for benefits under the act is DENIED.

A
DANIEL L. LELAND
Administrative Law Judge

DLL/es/lab

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this Decision and Order, by filing a notice of appeal with the ***Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601***. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington D.C. 20210.